

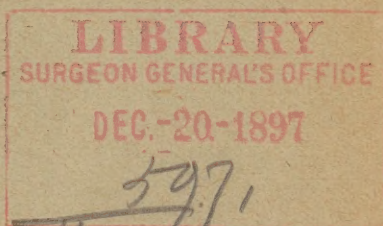
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GOUT AND RHEUMATISM AS FACTORS IN THE
ETIOLOGY OF GLAUCOMA.*

BY STEPHEN OLIN RICHEY, M. D.

WASHINGTON, D. C.



presented by the author

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GOUT AND RHEUMATISM AS FACTORS IN THE ETIOLOGY OF GLAUCOMA.*

BY STEPHEN OLIN RICHEY, M. D.

WASHINGTON, D. C.

Within the past year an eminent colleague said to me: "I think iridectomy in chronic glaucoma is indefensible; but what *is* glaucoma?" Agreeing with him in regard to operation in chronic glaucoma, it is yet with some diffidence that I essay this consideration of the malady.

The least confusing presentation of the subject is under its two extreme forms, acute inflammatory glaucoma, and chronic simple glaucoma. All other varieties occur between these two, partaking more or less of the one or the other, due to the same cause, in differing degrees of intensity; including even so-called secondary glaucoma, which may be *secondary* to keratectasia, or fistula of the cornea, in which affections the support to the blood vessels of the fundus is diminished by the subnormal intra-ocular tension in the same way as by a mydriatic; to irido-cyclitis, or retinal hemorrhage, dependent upon gout; occlusion of the pupil, luxation of the lens, intra-ocular tumors, choroiditis, myopia; any of them may cause the increase of tension of a pseudo-glaucoma, but not the intra-ocular tension characteristic of true glaucoma. Secondary glaucoma has a definite local exciting cause, but there is also a constitutional predisposition, as in glaucoma following cataract extraction,

Propositions.

First. Increased intra-ocular tension in glaucoma is caused chiefly by distension of the blood vessels internal to the sclerotic; rarely, and to a slight degree, by derangement of intra-ocular secretion, or excretion.

Second. Associated with increased intra-ocular tension is high general arterial tension, or arterio-sclerosis, and pulsation of the intra-ocular arteries and veins; with at least functional disturbance of the heart.

Third. As a result of persistent venous stasis hyper-

*Read May 4, 1897, in the Congress of American Physicians and Surgeons, at Washington, D. C.

plasia of connective tissue with substitution (substitutive fibrosis) takes place in all the tissues of the bulb.

Fourth. In chronic glaucoma the *materies peccans* of rheumatism cannot be excluded as a factor in causation; no reason appears for suspecting it of any agency in acute glaucoma,

Fifth. Every form of glaucoma is owing primarily to gout (or goutiness), or to acquired syphilis in its tertiary stage

Sixth. Iridectomy, the first violence of the attack being past, so reduces intra-ocular pressure as to permit the vena vorticosæ to be emptied, thus restoring the intra-ocular circulation and establishing normal tension. Any operation which lessens the surface pressure upon the vascular system within the eye, whatever the result, seems in reason open only to this explanation; for such vessels have no vaso-motor* system, and no power of contraction, but dilate and contract in response to the greater or less force of the extra-ocular blood current.

Intra-ocular tension in a normal eye is increased at every *pulse-beat*†, and at every expiration; and is lessened during inspiration. By experiment, retardation of the venous outflow increases pressure in the vitreous, and lowers it in the aqueous chamber; the venous outflow being normal, a reduced arterial supply lowers tension, and *per contra*, shewing the control of intra-ocular tension by the intra-ocular, depending upon the general, vascular condition. To the hypothesis of a derangement of the functions of secretion and excretion as a cause of high intra-ocular tension serious, if not insuperable objections exist, although it has been accepted for many years as the best offered. It is based upon an assumption that "increased intra-ocular tension is the essence of the disease," and the cause of all its evils, and that glaucoma is purely local. It is unsatisfactory to one who has watched

*Gray (Anatomy, 8th ed, page 77,) quotes Kolliker, "the majority of the arteries of the brain and spinal cord, and those of the choroids are unprovided with vaso-motors."

†The dependence of intra-ocular tension upon the extra-ocular arterial tension is shown by some late "Observations on the Retinal Blood-stream," (C. H. Usher, Oph. Rev. Dec. 1896.)

NOTE—"According to Klein and Svetlein the retinal vessels are not influenced by division or stimulation of the sympathetic." (Physiology, Landois and Sterling, 2d ed., page 625.) "Since the nervous supply of the choroid is especially distributed to the muscular tissue of the blood vessels the component *may be regarded* (?) as vaso-motor in character." (System of Dis. of the Eye, vol. 1, page 261, Norris & Oliver.)

an attack of acute glaucoma from its manifest inception, unless he be willing to ignore the constitutional history and complications of the malady; or to one who has followed the progress of a chronic glaucoma through all its phases and modifications, not limiting his observation to the special local developments. The disease has been correctly defined as "a complex of symptoms," for the involvement of each component tissue of the bulb compels it to give voice to its distress. Though high intra-ocular tension is a cause of some of the other symptoms, derangement of intra-ocular secretion could not accomplish what we find in glaucoma. If there were at the same time increased secretion and retention, the intra-ocular contents, by the action of both, would only the more quickly reach a point short of the high tension of acute glaucoma at which the resistance to secretion would be too great for its continuance; for the secretion of aqueous is a transudation, an osmosis, and lacks the intensity even of glandular secretion.

The term *secretion* offers no conception of energy.

Such an objection would seem to be fatal to the theory, though it is not claimed that secretion is not increased or retained in glaucoma; only that by this means a higher degree of intra-ocular tension *cannot* be produced than is to be found in serous iritis. It is a mere question of physics. That glaucoma has occurred in cases of aphakia, of aniridia, and in others in which the anterior filtration channels were found patulous would prove that impeded excretion had no agency in those instances. In view of such observations, when the anterior filtration channels are found closed in a dissection of an eye after the disease has run its course, we are not justified in assuming that the disease was due to impeded excretion.

Again, periods of increased intra-ocular tension occur, and pass off without mechanical interference, as in the prodromata. Could this be, if it were caused by closure of the anterior filtration channels?

The character of that obstruction is usually such, that once "the vicious circle" is formed, it can be broken only by foreign aid. Has the volume of the intra-ocular fluids (exclusive of the blood within the vessels) any important bearing upon the high intra-ocular tension of glaucoma?

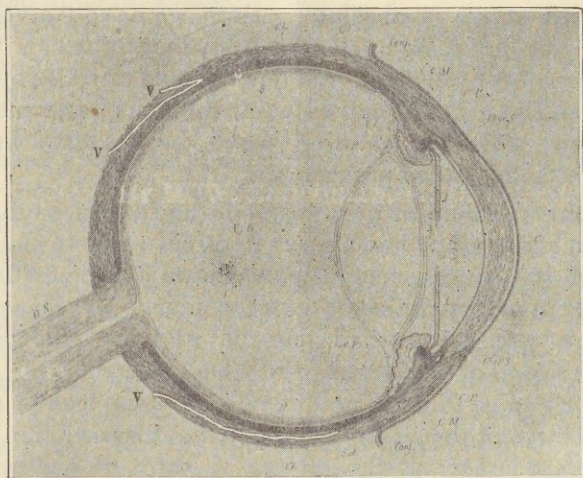
Advancement of the lens and iris is ordinarily observed in glaucoma, with no apparent pathological changes in

the vitreous; if the *volume* of the vitreous did vary, its consistency is such as to preclude the rapidity of variation necessary to account for the modifications of tension in the eye. An increased volume of aqueous humor in the eye cannot produce advancement of the lens and iris (much less of the corpus vitrei), it being *in front* of the lens, and able to distribute itself easily and equally on both sides of the iris, without pressure upon it. If the anterior channels of filtration were obstructed, the impaired outflow in this direction would increase the depth of the anterior chamber, as in serous iritis. When occlusion of the pupil occurs in plastic iritis, and the aqueous humor secreted behind the iris forces it forward in contact with the cornea, we do not promptly have glaucoma as a result. When the anterior filtration channels are closed by the iris, the lens and iris must *have* advanced, or the iris must *have been* dilated, by pressure from behind by a condition *already* present, which causes advancement of the vitreous, lens and iris, and increased intra-ocular tension. Also, when the aqueous is diminished by evacuation through a corneal incision, do not the lens and iris advance promptly to the posterior surface of the cornea? A normal volume of aqueous humor maintains a normal anterior chamber; an increased volume deepens, and a *reduced* volume of aqueous shoals the anterior chamber. Must not such an evacuation take place very slowly to avoid rupture of the distended blood vessels of the fundus by the sudden removal of the support in front to the *vis a tergo* of the general arterial pressure? It is an engorgement of the blood vessels of the fundus depending upon the pressure in the extra-ocular arterial vessels, upon the force of a bounding heart, which, *after iridectomy*, causes intra-ocular hemorrhage in the case of rotten vessel-coats; that, in some instances, prevents the restoration of the anterior chamber; that may end, if the general arterial pressure is sufficiently great and sustained, in malignant glaucoma.

The only rational explanation of a good result from iridectomy in glaucoma is, that a happy moment is chosen when the first force of the storm is spent, and the operation, by easing the pressure upon the intra-bulbar tissues and vessels, permits the static venous blood to move, and *active* circulation to be restored. This moment may be designedly secured, by first relieving general arterial tension

and quieting the action of the heart by a heart sedative; better, by HOT baths. To operate under other circumstances may mean an untoward result, and probably accounts for the difference in result of the same operation in similar cases. These comments necessarily apply to any operation which favors an advancement of the vitreous, lens, and iris.

Reasoning, directly and by exclusion, from frequently ascertained and unquestioned facts, constrains to the conclusion that a condition always found in acute glaucoma, which will account for all its features without distortion, offers the true explanation of this form of disease, viz :



A Vertical

~~Arterial~~ section of the eyeball from before backwards, enlarged five times. The white lines show the length of the channels of the vena vorticosæ ($1\frac{1}{2}$, $3\frac{1}{4}$, and $5\frac{1}{2}$ mm.) relative to the thickness of the sclerotica, which is not usually appreciated. The channels do not extend directly back, but backward and downward. The proper anatomical entrance and exit of the longest channel cannot be shown in the profile.

A great engorgement of the intra-ocular blood vessels, overfilling the space within the sclerotic, most so posteriorly, about the entrance of the arteria centralis, the twelve to twenty short ciliary arteries, and the two long ciliary arteries which pierce the sclerotic at a right angle; elongating the corpus vitrei in the direction of the length of the vessels by lateral pressure, which is magnified by the acute venous stasis developed by any slight increase of intra-ocular pres-

sure upon the vena vorticosæ as they pass very obliquely out through the sclerotic; and advancing the corpus vitrei, lens, and iris, by pressure from behind, reducing the depth of the anterior chamber, and applying the iris to the cornea; thus obstructing the anterior channels of filtration, after increased intra-ocular tension has been produced.*

The *direct* course of the arteries through the sclerotic permits ready access of arterial blood, any increase of which, beyond what is normal within the eye, *must* exert some pressure upon, and stretching of the sclerotic, which in turn narrows the lumen of the veins passing *obliquely* through it, still further increasing the intra-ocular pressure until stasis is absolute. Here we have “a vicious circle,” but in a region different from that in which it has heretofore been located, and one capable of relief by the control of the primary extra-ocular influence, and without operation; which we so often see to be the case in the earlier attacks, called prodromata.

The engorgement of the vessels of the fundus oculi, existing in acute glaucoma always in direct ratio to the violence of the attack, conclusively shows it to be distinctly vascular in origin, which is extra-ocular. By reason of the high general arterial tension, and the quickened, forcible action of the heart, the blood entering the eye, posteriorly most directly and in most volume, makes room for itself within the sclerotic by driving forward the solid moveable contents of the globe. A state of the system which leads to a reduction in the caliber of the peripheral arterial vessels, demanding an increased effort on the part of the heart, will account for this; because, as the contraction of the peripheral vessels diminishes the size of the blood current passing through them, the propulsive efforts of the heart must increase, and these determine the volume of blood in the direction of *least* resistance. Therefore, the vessels which receive it are the intra-cranial vessels which have no vaso-motor constrictors, and those of the internal organs whose temperature is higher than the body-surface. Obviously, the cause must be some element in the blood which narrows the caliber of the vessels by irritation, aided at times by the contracting influence of low surface temperature, or by some emotion exciting

*“Making channels from $1\frac{1}{2}$ to $5\frac{1}{2}$ mm. in length” (Metz, Anatomy and Histology of the Eye, 1868, page 35.

a reflex from the base of the brain, or creating tumult of the heart.

Whether the intra-ocular vascular engorgement is ever excited by the precipitation of sodium bi-urate within the eye, or not, will probably be settled only by accidental observation. I think, not necessarily. It is more than probable that great emotion disturbing the regularity and stability of the heart's action, or a fit of indigestion with distension of the stomach and consequent reduction of the peri-cardiac space, when the heart is already laboring to overcome the resistance offered by the narrowed peripheral vessels to the passage of a normal blood-current, is the first step in acute action.

In which event, the first indication is to relieve the heart of excessive work by dilating the peripheral vessels, which at the same time will divert the pressure of the blood column from the intra-cranial vessels, from the intra-ocular vessels, and thus reduce intra-ocular tension.

Chronic Simple Glaucoma differs so much from the other forms of the disease, and especially from acute inflammatory glaucoma, that some observers have doubted if it be glaucoma at all. It is glaucoma, a chronic interstitial ophthalmitis; a most perfect presentment of a chronic process which can be imagined, and is brought about by the same initial influences which cause the acute form, acting without explosive force. As acute glaucoma is vascular and violent, this form is interstitial and slowly progressive. For years, its development is preceded by a stationary, or an imperceptibly increasing, general arterial tension, united with palpitating and uncertain action of the heart, and increasing rigidity of the sclerotic coat, leading to the deduction that senility is an etiological factor. Age is a predisposing influence, as it is physiologically a period of substitutive fibrosis; if it were an active agent, we would find an increasing percentage of simple glaucoma with advancing age. A new connective tissue growth has, by anatomical dissections and microscopical examinations, been found in all the structures of the glaucomatous globe, and as a formative predominance of special tissue arises in specialized irritability of such tissue, for its cause we must look to some irritant exciting vascular and neurotic disturbances affecting nutrition. The presence of such an irritant, inherited or acquired, constitutes a *diathesis*. The preponderance of evidence,

(ocular tissue changes and symptoms, and the associated constitutional manifestations) indicate glaucoma to belong to acquired tertiary syphilis (rarely); or to the diathesis termed *arthritic*, which includes gout and rheumatism.

These are congeners in their origin (inadequate digestion) and in their pathological expression — the modification and proliferation of fibrous tissue, and the irritation of serous membranes.

Gout and Rheumatism.

The two important factors in the arthritic diathesis, generated in the system and constant, are organic acids; uric acid, a product of inefficient proteid metabolism; in excess (“largely a renal question” (Ewart,)) the cause of gout; and lactic acid, a fermentation product of the hydro-carbons (sugar); in excess (due to the condition of the integument), the cause of rheumatism. No uric acid has ever been found in the blood in rheumatism, and its excretion by the urine in this disease does not exceed the quantity usual in fevers. Haig believes both affections to be due to uric acid, to which Garrod and others demur. In acute attacks of both diseases there is chill, fever, and sweat, pain in the joints, a coated tongue, loss of appetite, thirst, constipated bowels, and scanty, high-colored urine which makes a red deposit on cooling (most of which is a familiar picture in acute inflammatory glaucoma.) In rheumatism the fever is more marked, the sweat more profuse, and acid (containing lactic acid,) metastasis more common, and the joints seized in the first attack are larger for even the peri and endo-cardium, the pleura, the peritoneum, the spinal or cerebral meninges may be involved; for lactic acid is excreted by the urine under no conditions, beyond an approximately fixed quantity, is not precipitated, no deposit ever having been found; nor is it known to combine tenaciously with any constituent of the blood. When in excess, therefore, *unless excreted by the skin*, it is retained in the circulation and acts as an excitant; an *excitant*, for Gaskel found that when dilute lactic acid was injected into the circulation of a frog the caliber of the arterial vessels was enlarged and the ventricle of the heart was relaxed. The action of dilute alkaline solutions upon the heart and the arterial coats was the reverse; contraction.

Lactic acid, denied passage through the skin by the

constraining effect of cold and damp upon the dermal vessels, passes with the blood in greater volume to the deeper-seated organs of higher temperature, and excites violent action there. The larger the joint and the higher its temperature, the larger the volume of blood, consequently the greater the quantity of lactic acid which *will pass through it*, and the greater the stimulation. The lower temperature of the small joints, and the smaller volume and slower current of blood circulating through them, limits their exposure to the effect of lactic acid. The great excess of lactic acid in the blood in this disease, its saturation of the saliva, the sour (lactic acid) sweats, the purely conjectural attitude of the bacterial and the neurotic theories, and the suggestive habits and environment of rheumatics, continue to the lactic acid theory the prominence and probability which have been attached to it for some years.

These are not diminished even by the failure of its treatment by alkalies, for, according to Sir W. Roberts, it is difficult to influence the reaction of the blood, requiring large quantities of acids or alkalies, and then the result is uncertain and temporary, owing to their prompt elimination by the urine. Moreover, "lactic acid is *formed* whenever sugar in solution is brought in contact with an alkaline, or earthy, carbonates in the presence of a special ferment, as casein." (U. S. Dispensatory, 1894, page 66.) An excessive formation of lactic acid probably arises from the inefficient preparation in the mouth of hydro-carbons, due to insufficient salivation, a subnormal percentage of ptyalin in the saliva, or the unfavorable conditions to which they are subjected (hot and cold drinks with food); for ptyalin acts only between 95° and 102° F. A low ratio of ptyalin, which the analysis of the saliva of some rheumatics has shown, taken with an inherited delicacy of the skin, may account for an inherited tendency to rheumatism.

The claim in favor of dampness and cold as initial causes, because rheumatism is more common in New England than elsewhere in the United States, and in Scotland and Ireland, than elsewhere in Great Britain, happens to be coincident with the fact that in those sections the hydro-carbons, eaten in a manner least favorable to ptyalization, are a staple food.

The writer has dwelt at such length upon this subject,

because rheumatism has so often been included with the uric acid diseases, which, to his mind, is clearly an error; because it is a part of the subject assigned to him; and because in consequence of the excess of fibrine (sometimes twice the normal) in the blood in rheumatism, the affections of the eye due to it are apt to be *adhesive* in character. Plastic, or parenchymatous iritis may be due to rheumatism; serous iritis is of gouty origin.

Gout in its *articular* manifestations is a local affection of constitutional origin; abarticular gout, latent or declared, precedes the seizure of a joint, and the term gout should include all the baneful activities of uric acid in whatever part of the body they are met. Although in addition to the very chronic process of fibrosis which is the tissue-form ultimatum of malnutrition, and due in part to an excess of urates in the circulation, a common feature of gout is the local deposit of sodium bi-urate, which may be frequently seen in the palpebral and bulbar conjunctiva, in the skin as tophi, as well as in the joints, and as Meyer (Fergus's Transl. Dis. of the Eye, page 248) states, "in acute glaucoma the aqueous is muddy and sometimes forms deposits on the posterior surface of the cornea," which condition I have not observed. It is not easy to determine the character of such deposits, but I have held deposits in this situation following serous iritis under suspicion of being sodium bi-urate, though Garrod claims that the inflammation excited by their presence destroys such deposits; we do not find this true in keratitis punctata. As a deposit within the eye in sufficient quantity to be seen is not essential to its power to create excitement in so delicate a structure; as only irritation sufficient to fix the focus of vascularity when the heart is in exalted action from disturbed emotion, etc., is required to decide an acute glaucoma; a determination of the presence of such a precipitate would be inconclusive. Such investigation is beset by invincible difficulties. The urates, while remaining in the circulation, produce irritation of the fibrous tissues and serous membranes with which they come in contact, and may thus decide a region for the concentration of any general vascular tumult, but they do not directly cause *acute* local trouble unless massed by precipitation, which is favored by the lower temperature of the small joints, their consequent comparatively sluggish circulation, their composition of fibrous and serous tissue for which the

materies morbi of gout has a predilection, and the injuries to which they are exposed. All of these conditions the globe of the eye satisfies.

The presence of a precipitate in the bulb not being a *sine qua non* to an acute glaucoma, an excess of urates in the blood and palpitating action of the heart being sufficient to declare such an effect, we may accept as evidence of gout the existence of migraine, eczema, uncertainty of temper with insomnia, mental depression or hebetude, crackling of the cervical spine, pain in the gastrocnemius after a short walk; certain forms of scleritis, keratitis, and conjunctivitis, serous iritis, pulsating arteries and veins, increase of albumen in the aqueous, extravasations of venous blood, turgid and swollen ciliary processes, and great œdema; pharyngitis, cystitis, so-called idiopathic urethritis, herpes, or progressive deafness. The longitudinal ridges and broad lateral furrows, and the extreme brittleness of the finger nails, are additional indications of the degenerating influence of the diathesis. The thread-test of the blood for uric acid, however, is crucial.

Gout expressing itself in glaucoma has not necessarily other serious manifestations. I have never seen a glaucomatous person with a bald head.

The malaise and slight chilliness, followed by flushing of the face and mental stupor, so often observed in uric-acid-ædemia, is not rarely mistaken for some form of malarial fever, as it may have periods of remission and recurrence within the twenty-four hours, to which error the beneficial effect of quinine, excluding the urates from the circulation, is contributory. An examination of the blood for the malarial parasite, or uric acid, may be essential to a diagnosis.

That no deposits of sodium bi-urate have been externally manifest does not justify the exclusion of gout as a cause of glaucoma, for gouty changes sufficient to destroy life itself have occurred without apparent joint deposits or active inflammatory processes.

The known incidents of acute podagra and acute glaucoma, and their perfect mimicry, allowing for the function of the organs attacked, show each to be an "uric acid storm," the region being selected by reason of its individual specialized irritability, a tendency ordinarily inherited.

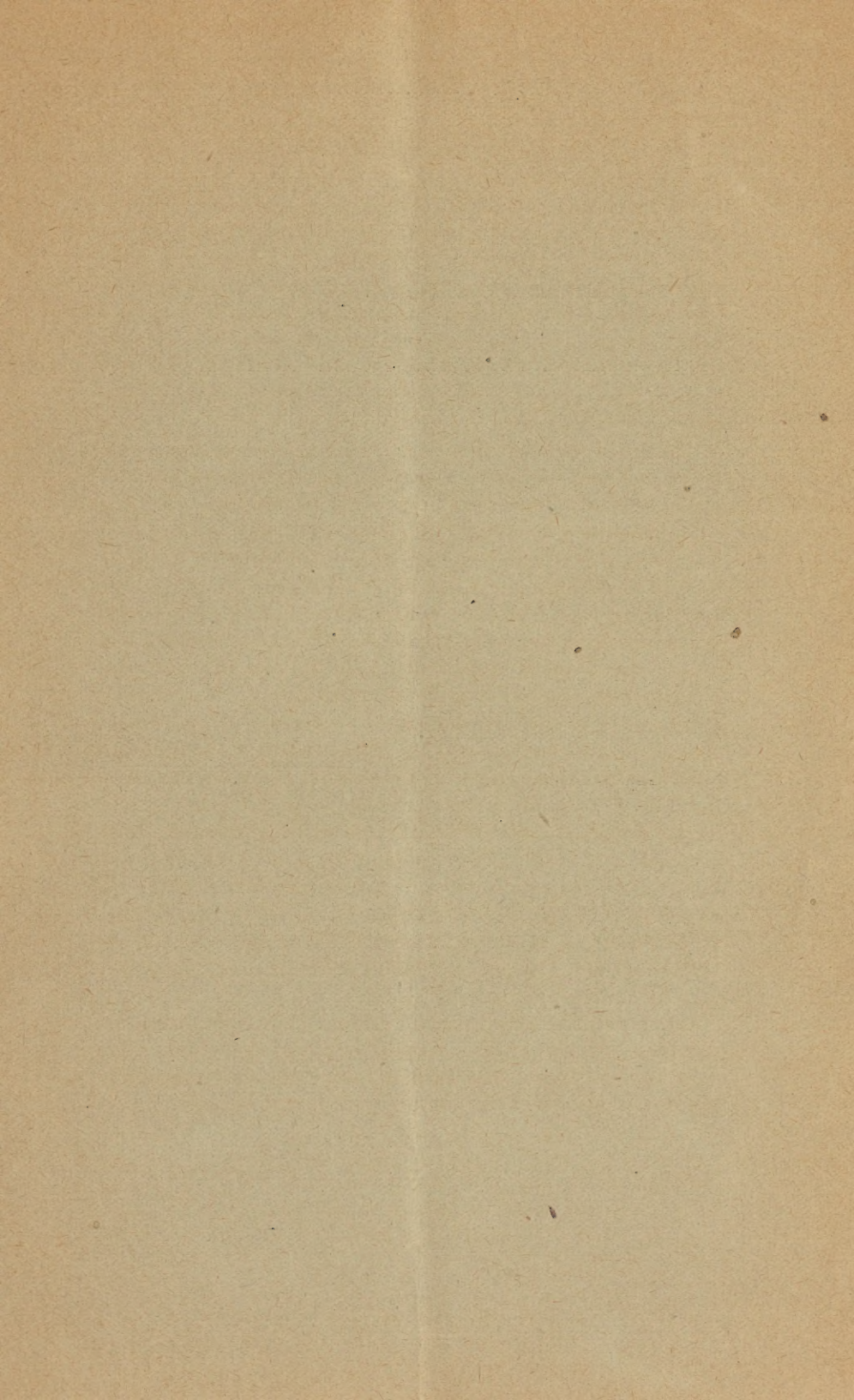
Sir William Roberts (Croonian Lectures, 1892, Uric Acid, Gravel, and Gout, pp. 33 and 119,) offers a very

fascinating suggestion in explanation of habitual excess of uric acid in a system.

“Structurally, no essential difference exists between the kidneys of birds and snakes, and those of the highest order of mammals—man. Functionally, there has been immense evolution from the solid or semi-solid form to the watery form of urine, and in the substitution of a soluble form of nitrogen (urea) for one very sparingly soluble (uric acid). This particular of physiological evolution has not yet been perfected, and man must still excrete 8 or 10 grains of uric acid a day.

“In the course of such an evolution a double change would be in progress, namely: A gradual substitution of urea for uric acid as the final term of proteid metabolism; and the complementary modification in the functional aptitudes of the renal cells—that is to say, an ever-increasing aptitude for the excretion of urea, and an ever-diminishing aptitude for the excretion of uric acid. When, after long æons, the mammalian type was at length evolved, there remained only a small residuum of uric acid to be dealt with, and, correspondingly, only a small residuum of the original aptitude of the renal cells for its excretion. A feature thus begotten would present great variation in different individuals. Generators of gout, and members of gouty families, are, for the most part, people of fine frames and ample appetites, who are withal great consumers of nitrogenized food. In such persons there is an enhanced production of urea which expands by grams, while uric acid expands only by grains. There would thus arise a persistent demand on the urea excreting faculty which could only be satisfied by an encroachment on the residuary faculty for the excretion of uric acid.”

When he produces urea and no uric acid from nitrogenous food; and when from this he has assurance that “he eats to live,” and not to die, man will probably have reached his physical millenium, and have no glaucoma.



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